

A Proposed Concept Model for Cancer Risk in Nigerian Electronic Waste Exposure – A Brief Report

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ABSTRACT

Aim: This report aims to render a proposed concept model for cancer risk in Nigerian electronic waste exposure by making deductions from data on the assessment of Nigerians' exposure to toxic metals in e-waste, using biomarkers of exposure and genotoxicity to evaluate the risk of cancer development.

Materials and methods: In the cross-sectional study, 632 consenting participants, consisting of 381 e-waste workers (EW) and 120 environmental e-waste exposed participants (EEEEP), age-matched with 131 unexposed participants (controls), were enrolled from Benin, Lagos and Ibadan, Southwestern Nigeria. Levels of selected toxic metals in blood and essential metals in serum were determined using inductively coupled plasma-mass spectrometry. Oxidative stress biomarkers, including malondialdehyde and uric acid (UA), and activities of enzymatic antioxidants [catalase, superoxide dismutase (SOD), γ -glutamyltransferase (GGT) and glutathione peroxidase (GPx)], were determined in serum using standard methods like spectrophotometry. Genotoxicity biomarkers – wild-type tumour suppressor protein (wt-p53), 8-oxoguanine-DNA glycosylase (OGG1), and 8-hydroxy-2'-deoxyguanosine (8-OHdG); glutathione

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(GSH); and tumour markers [prostate-specific antigen (PSA) and alpha-fetoprotein] – were determined in serum using ELISA. Micronucleus assay was carried out using microscopy. Data were analysed using ANOVA and Pearson's correlation coefficient at $\alpha 0.05$.

Results: There was evidence indicating elevated levels of genotoxic toxic metals, decreased levels of genome protective metals, increased oxidative stress markers as well as reduced cellular antioxidants in both EW and EEEP compared to controls. Additionally, the levels of wt-p53 in EW and EEEP were lower than controls, while OGG1 activity in EEEP was higher. The PSA and alpha-fetoprotein in EW were more elevated than EEEP and controls, respectively. The MnPCE/1000PCE in EW was higher than EEEP and controls.

Conclusion: The proposed schematic model could be adopted to illustrate cancer risk in Nigerian population exposed to electronic waste.

Keywords: cancer risk, model, e-waste, genotoxicity, metals.

INTRODUCTION

The scourge of electronic waste (e-waste) is currently a major global concern, particularly in developing countries like Nigeria, where reprocessing is substantial but unregulated (1-5). The possible contribution of e-waste toxic metals to the risk of cancer development in Nigeria has received little attention. This brief report aims to render a proposed concept model for cancer risk in Nigerian e-waste exposure by assessing the exposure of Nigerians to toxic metals in e-waste, using biomarkers of exposure and genotoxicity to evaluate the risk of cancer development. □

MATERIALS AND METHODS

In the present cross-sectional study, 632 consenting participants, consisting of 381 e-waste workers (EW) and 120 environmental e-waste exposed participants (EEEP), age-matched with 131 unexposed participants (controls), were enrolled from Benin, Lagos and Ibadan, Southwestern Nigeria.

Levels of toxic metals (cadmium, lead, mercury, arsenic, chromium, nickel, molybdenum, aluminium, vanadium, thallium, antimony and tin) in blood and essential metals (zinc, selenium, copper and cobalt) in serum were determined using inductively coupled plasma-mass spectrometry.

Vitamins A, C and E, oxidative stress biomarkers – malondialdehyde and uric acid (UA), and activities of enzymatic antioxidants [catalase, superoxide dismutase (SOD), γ -glutamyltransferase (GGT) and glutathione peroxidase (GPx)] – were determined in serum using standard methods like spectrophotometry.

Genotoxicity biomarkers – [wild-type tumour suppressor protein (wt-p53), 8-oxoguanine-DNA

glycosylase (OGG1), and 8-hydroxy-2'-deoxyguanosine (8-OHdG)]; glutathione (GSH); and tumour markers [prostate-specific antigen (PSA) and alpha-fetoprotein] – were determined in serum using ELISA. Micronucleus assay was carried out using microscopy. Data were analysed using ANOVA and Pearson's correlation coefficient at $\alpha 0.05$. □

RESULTS

There was evidence indicating elevated levels of genotoxic toxic metals, decreased levels of genome protective metals, increased oxidative stress markers as well as reduced cellular antioxidants in both EW and EEEP compared to controls. Additionally, the levels of wt-p53 in EW and EEEP were lower than controls, while OGG1 activity in EEEP was higher. The levels of PSA and alpha-fetoprotein in EW were more elevated than EEEP and controls, respectively. The MnPCE/1 000 PCE in EW was higher than EEEP and control. The proposed concept model for cancer risk in e-waste exposure (Figure 1) was developed based on data, deductions and inferences derived from the results of the present study.

The development of biological models or illustrative schemas are useful means of facilitating the assessment of research data or results. Modelling has been reported to provide the kind of intellectual frameworks needed to transform data into knowledge (6). The proposed model in question in this study was developed to summarize the biological events that could lead to cancer susceptibility in Nigerian populations exposed to major e-waste. As seen in the proposed schema, the over 1 000 constituents (chemicals and metals) of e-waste (1) are indicated in the

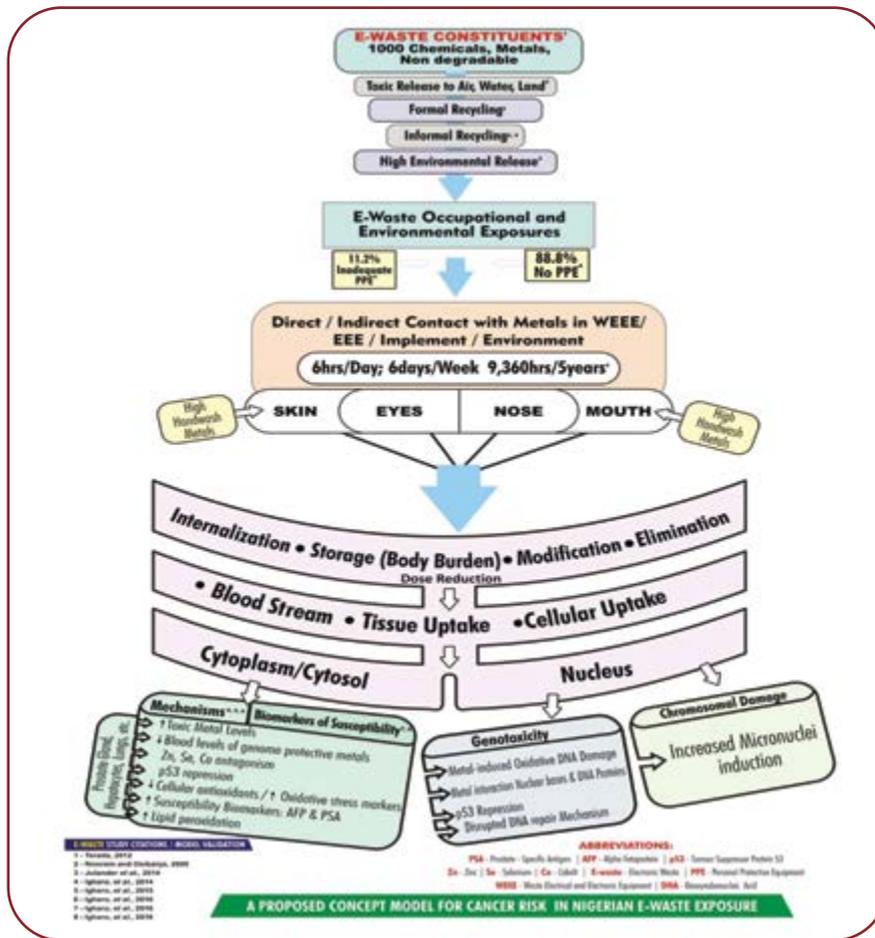


FIGURE 1. A proposed concept model for cancer risk in Nigeria e-waste exposure



FIGURE 2. Sections illustrating: (2a) a WEEE worker carrying out daily work routine without standard personal protective equipment; and (2b) a pictorial depiction of indiscriminate WEEE disposal practice that leads to high environmental contamination with e-waste-borne toxicants, observed in New Benin market area, Benin City, Nigeria

topmost part as presented. Due to informal recycling, there are toxic releases of these e-waste-borne constituents into the environment through the air, water and land, indicating a high level of contamination (3, 7, 8).

With the established crude informal reprocessing of e-waste materials in Nigeria (1, 8), de-

icted in Figure 2a, and the indiscriminate WEEE disposal practices observed in the e-waste high impact areas in Nigeria (depicted in Figure 2b during the study), it is notable that those practices are environmentally unfriendly and occupationally unsafe. Typically, workers were found with inadequate or no personal protective equip-

ment (PPE) and they disposed waste without adherence to safety standards. With these practices, it was seen that the Nigerian e-waste workers who represented the population of the present study were continuously predisposed to toxicant exposure on an average six hours per day and six days *per week*, totalizing an estimated number of 9 360 hours of exposure for every five-year duration on the job.

Body contacts (hands, skin, eyes, nose and mouth) were identifiable means of entrance of the e-waste-borne substances into the body, resulting in a high body burden, which the body attempts to handle via the mechanisms of biochemical modification and elimination (which may be partial) in order to achieve dose reduction as shown in the proposed model. After a possible dose reduction/modification, the resultant toxicant found in the blood stream may be taken up by tissues and cells, and eventually find their ways to the cytoplasm/cytosolic compartments as well as the nucleus.

The presence of toxicants and the resultant damage in target organs and tissues, such as prostate glands, hepatocytes, lungs etc. has been indicated by the reported levels of biomarkers of

genotoxicity and cancer susceptibility (8-13), as indicated in the terminal part of the proposed concept model. □

CONCLUSION

The proposed schematic model could be adopted to illustrate the risk of genotoxicity and cancer susceptibility in Nigerian populations exposed to electronic waste. □

Conflicts of interest: none declared.

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